

ment was made at the half-moon, when the moon was high up, so that the light fell obliquely on the window and did not shine directly on the selenium.

On throwing the moonlight on the selenium by means of a plane mirror, the needle was at once deflected 20 divisions of the scale; on placing the mirror outside the window so as to send the moonlight perpendicularly through the window on the selenium, the deflection of the needle was 40 divisions. The window was kept closed during these experiments.

On another evening when the moon shone very obliquely on the window, and the selenium was exposed on the inside of the window directly to the moonlight, the needle was deflected 100 divisions of the scale, and the deflection increased to 150 divisions after exposure for about 3 minutes.

The change in the resistance of the selenium was from 60,000 to 70,000 ohms.

These experiments show that the action on the selenium is due principally, if not entirely, to radiations belonging to the visible part of the spectrum. Light rays of all kinds, particularly the greenish yellow, produce an instantaneous effect followed by a more or less gradual effect, which continues to increase during exposure for several minutes.

These facts suggest two hypotheses as possible explanations, which may help as guides in further experiments, but which cannot be accepted as proved without further evidence.

(1) That the light falling on the selenium causes an electromotive force in it, which opposes a battery-current passing through it, the effect being similar to the effect due to polarization in an electrolyte.

(2) That the light falling on the selenium causes a change on its surface akin to the change which it produces on the surface of a phosphorescent body, and that in consequence of this change the electric current is enabled to pass more readily over the surface of the selenium.

XIII. "On the Production of Glycosuria by the Effect of oxygenated Blood upon the Liver." By F. W. PAVY, M.D., F.R.S. Received June 17, 1875.

In a communication on "Lesions of the Nervous System producing Diabetes," presented to the Royal Society in 1859 (*Proc. Roy. Soc.* vol. x. 1859-60), I made known that division of certain parts of the sympathetic system occasioned the presence of sugar in the urine. The effect of puncturing the floor of the fourth ventricle (Bernard's celebrated experiment) had been for some time previously familiar to physiologists; but nothing had been ascertained about the production of diabetes by lesions of the sympathetic, until my experiments upon the subject were conducted; and it was in attempting to discover the channel through

which the medulla oblongata influenced the liver, that I was led to recognize the facts which my researches disclosed.

These results merely showed that there were other means besides puncturing the floor of the fourth ventricle by which artificial diabetes could be induced. They did not explain the reason of the appearance of sugar, and I still sought to discover something upon this point. Failing to reconcile any explanation that has been suggested with the evidence furnished by experiment, I have from time to time pushed inquiry in various directions, but always with a fruitless issue, until the summer of 1874, when I came across the results which it is the object of this communication to make known, a brief announcement of them having been previously made in a letter to the Secretaries of the Royal Society shortly after the close of the session of last year.

For some time past I have been led to look to an altered condition of the blood flowing to the liver as likely to prove the most probable cause of the transformation of amyloid substance into sugar, which evidently constitutes the foundation of the artificial diabetes following operations upon the nervous system. Schiff is of this view, and (*'Journal de l'Anatomie et de la Physiologie,' Paris, 1866*) has referred the escape of sugar from the liver, and thence the production of glycosuria, to the development of a ferment in the blood as a result of the hyperæmia which follows the operations on the nervous system which occasion artificial diabetes. It is not necessary, according to his view, that there should be hyperæmia specially of the liver; but hyperæmia anywhere may lead to the development of the ferment which he alleges to be the source of the appearance of sugar. "*Le diabète par suite de l'hyperémie pourrait donc bien ne pas être l'effet spécifique d'une hyperémie du foie, mais de chaque hyperémie générale d'une certaine étendue.*" I have carefully examined this view of Schiff, and cannot obtain evidence of the development of a ferment in the manner asserted; moreover I have tried the effect of introducing a secretion, viz. saliva, into the circulatory system, which is known to act as an energetic ferment upon the amyloid substance of the liver. At first I introduced it into the general circulation through the jugular vein, and failed to observe the production of saccharine urine. Later on, thinking the experiment might be more effective, I injected it into a branch of the mesenteric vein, so that it would all pass directly to the liver. Upon one occasion I found that the urine, from some cause or other, became to a moderate extent saccharine; but in a large number of other experiments the operation was attended with a negative result. The quantity used varied in different instances, the largest amount employed being 13 fluid drachms. In some cases the saliva was introduced in a pure state, in others after dilution with varying proportions of water.

Looking at the fact that the amyloid substance is contained in the hepatic cells, it is scarcely surprising that a ferment of a colloid nature

contained in the blood should fail to be capable of determining the production of sugar whilst passing through the vessels of the liver.

Having so far proceeded without success, it now occurred to me to try the effect of introducing defibrinated arterial blood into the portal system. I was led to experiment in this way from having a long time previously observed that when arterial blood only was allowed to flow through the liver (as, for instance, when the portal vein was tied and the hepatic artery left free), sugar escaped from the organ to such an extent as to render the contents of the circulatory system strongly saccharine. This result I had commented upon as being somewhat surprising, and as furnishing evidence standing in opposition to Bernard's glycogenic theory. I had not succeeded by the operation in producing glycosuria, because, as it appeared to me, no urine was secreted, owing to the ligation of the portal vein leading to such a diversion of blood from the general circulation, by the accumulation occurring in the portal system, that the flow through the kidney was too slight to allow of it. I had endeavoured to overcome this obstacle by connecting, through the medium of a canula, the portal with the right renal vein after ligaturing the corresponding renal artery. If the experiment had succeeded, the liver would have been left with its arterial supply, but the portal stream would have been diverted and made to reach the inferior cava without traversing the hepatic vessels. As regards the operation, this I found I could accomplish; but each time I performed the experiment the object I had in view was frustrated by the canula becoming quickly filled with a plug of blood-clot. It was whilst under this difficulty that I thought of collecting blood from an artery, defibrinating it, and then introducing it into the portal system. I had considered it possible that some slight effect might be perceptible, but had not anticipated the strongly marked result which is producible.

The amount of blood used has been from 10 to 18 fluid ounces. After the production of anæsthesia by chloroform the blood was collected from the carotid artery, stirred in order to defibrinate it, strained, and then very slowly injected into a branch of the mesenteric vein. In one experiment, where half an hour had been employed in making the injection, the urine, at the completion of the operation, contained a notable amount of sugar, and half an hour later showed, by analysis, the presence of 15 grains to the fluid ounce. In a second the urine contained 10 and in a third 14 grains to the fluid ounce when collected three quarters of an hour after the operation.

The experiments were performed upon dogs, and in each case it had been ascertained that the urine was devoid of sugar before the operation.

It will thus be seen that these results leave no doubt about the decided production of glycosuria. The effect was not only rapid but of a strongly marked character. It is necessary, however, before concluding that the glycosuria was really attributable to the influence of the oxygenated blood, to have evidence that in the absence of oxygenated blood a

negative result would be obtained. I took care, by slowness of injection, to avoid allowing the circulation through the liver to be influenced by the effect of increased pressure within the portal system. I am not aware, and do not think from my previous experience, that any fallacy would be likely to arise from such a source; but I nevertheless deemed it advisable to exclude the possibility of its occurrence.

Having noticed the effect which has been described from the injection of oxygenated blood into the portal system, it became necessary to ascertain positively that it was attributable to the oxygenated condition of the blood, and not to any other cause. To decide this point an appeal to the counterpart experiment was made. Defibrinated venous instead of arterial blood was injected into a branch of the mesenteric vein; and upon each occasion where such an operation has been performed a negative result has been obtained. With the evidence thus furnished the conclusion may be warrantably drawn, that oxygenated blood in some manner influences the liver, so as to lead to the production of glycosuria. It may be inferred that, contrary to the effect of venous blood under a natural state of the circulation, it promotes the transformation of amyloid substance into sugar.

In performing these experiments of injecting defibrinated blood into the portal system, I came across an effect which I had not anticipated, and which upon several occasions frustrated the object I had in view. Defibrinated blood, as experimental physiologists know, may be injected into the veins of the general system without producing any special effect. Injected, however, into the portal system, it frequently gave rise to a complete stoppage of the portal circulation by inducing coagulation of the blood in the trunk of the portal vein and its ramifications in the liver. I merely mention the fact in this place, and am not aware that it has hitherto been noticed; but it may hereafter be found of interest in connexion with the subject of the coagulation of blood. Finding that several of my experiments proved fruitless from this cause, I tried means to obviate coagulation occurring, and amongst these was extirpation of the spleen. With this operation, as far as my experience has yet gone, the coagulation is prevented; and, moreover, I have noticed that the blood in the general system after death fails to possess its ordinary coagulating property.

The suggestion naturally occurs, that what has been stated in this communication affords an explanation of the glycosuria occurring after Bernard's puncture of the fourth ventricle and the various lesions of the sympathetic. Without any new agent being called in, sufficient is presented in the state of the blood to account for the production of sugar that occurs. By a vaso-motor paralysis affecting the vessels of the chylipoietic viscera the blood will reach the portal system without having become dearterialized in its natural way; and in this state it has been shown to possess the property of acting within the liver in such a manner as to determine the production of glycosuria.